Effect of Lecithin on CFA Induced Rheumatoid Arthritis in Female Wistar Rats

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Abstract - Rheumatoid arthritis (RA) is a chronic systemic autoimmune inflammatory disease that affects mainly the small joints of the hands and feet. RA is one of the most common inflammatory joint diseases and causes premature mortality, disability and compromised quality of life. The overall worldwide prevalence is 0.8% and steadily increases to 5% in women over the age of 70. RA is two to three times more common in women compared to men. In India the prevalence has been estimated to be 0.7%. Accordingly the main objective of the present study was to evaluate the activity of lecithin using in-vivo model, on CFA induced rheumatoid arthritis in Wistar rats of weights 250-260 g were selected & divided into 6 groups each of 6 randomly paw edema. The test and standard doses were administered orally. The paw edema was measured by using vernier calipers. At 21st day the animal blood collected through retro orbital route and haematological parameters were estimated. After the induction of CFA, the animal develops inflammatory responses and shows animals sacrificed by giving high dose of anesthesia then left hind paw was dissected and performed histopathology studies. The observations such as paw volumes recorded on 1st, 4th, 8th, 14th, and 21st days.

1. INTRODUCTION

Rheumatoid arthritis (RA) is a chronic systemic autoimmune inflammatory disease that affects mainly the small joints of the hands and feet. RA is one of the most common inflammatory joint diseases and causes premature mortality, disability and compromised quality of life[1]. RA is widely prevalent throughout the world. The overall worldwide prevalence is 0.8% and steadily increases to 5% in women over the age of 70. RA is two to three times more common in women compared to men. In India the prevalence has been estimated to be 0.7% [1].

The potential of the synovial inflammation to cause cartilage damage and bone erosions and subsequent changes in joint integrity is the hallmark of the disease. Environment –gene interactions described in the test promote loss of tolerance to self –proteins that contain a citrulline residue which is generated by post –translational modification. This anticitrulline response can be detected in T-cell and B-cell compartments and is probably initiated in secondarily lymphoid tissue or bone marrow. Thereafter, localization of the inflammatory response occurs in the joint by virtue of poorly understood mechanisms that probably involve micro vascular, neurologic, biochemical, or other tissue specific pathways. Synovitis is initiated and perpetuated by positive feedback loops and in turn promotes systemic disorders that make up the syndrome of the rheumatoid arthritis properties ACPA denoted anti-citrullinated protein antibody, and RF (rheumatoid factor).

The co-stimulation dependent interactions among dendritic cells, T-cells, and B-cells are shown as occurring primarily in the lymph node; these events generate an autoimmune response to anticitrulline -containing self proteins .In the synovial membrane and adjacent bone marrow, adaptive and innate,
immune pathways integrate to promote tissue remodeling and damage.

Positive feedback loops mediated by the interactions shown among leukocytes, synovial fibroblasts, chondrocytes and osteoclasts, together with the molecular products of damage; drive the chronic phase in the pathogenesis of rheumatoid arthritis. ADAMTS denotes a disintegrin and metallopeptase with thrombospondin-1,-like domains. DAMP damage associated molecular pattern, DKK-1, dickkopf-1, FcR Fc receptor, FcERI high-affinity IgE receptor, FGF fibroblast growth factor, GM-CSF granulocyte-macrophage colony-stimulating factor, HA hyaluronan, HSP heat shock protein, IFN-α/β interferon, MMP matrix metalloproteinase, NLR nucleotide-binding oligomerization domain like receptor, PAMP pathogen – associated molecular pattern, PAR2 protease – activated receptor, 2, PDGF platelet- derived growth factor, RANKL receptor activator of nuclear factor κB ligand, TGF-β transforming growth factor β, Th0 type T cell, Th1 type 1 helper T cell, Th17 helper T cell, TLR toll like receptor, TGFα tumor necrosis factor α and VEGF vascular endothelial growth factor.

Inflammatory mediators including cytokines, immune complexes and altered lipid metabolism circulate to promote several coexisting conditions in patients with rheumatoid arthritis. The primary goals in the treatment of RA are to control inflammation and slow or stop disease progression. Initial therapeutic approaches relied on disease-modifying anti-rheumatic drugs, or DMARDS, such as MTX and sulphasalazine [3, 5, 6]. These oral drugs work primarily to suppress the immune system and, while effective in this regard, the suppression of the immune system leads to an increased risk of infections. These drugs are also associated with side effects including nausea, abdominal pain, and serious lung and liver toxicities. Further, because these drugs often take an average of 6–12 weeks to take effect, rheumatologists may also couple them with over-the-counter pain medications or non-steroidal anti-inflammatories to treat the pain and inflammation.

Despite these shortcomings, DMARDS are still considered first-line therapies [7, 8]. Because of these harmful effects new compounds should be invented with high efficiency and low toxicity. Lecithin which is considered first in the treatment of rheumatoid arthritis using biochemical parameters like haemoglobin content, WBC and biochemical parameters like haemoglobin content, WBC and hematocrit percentage, statistical Analysis of result.

2. MATERIAL AND METHODS

Indomethacin & Lecithin were bought from local vendor, Vernier Calipers we utilized from our college laboratory.

2.1 Procedure

Wistar rats of either sex of age 2-3 months, weighing 250-270 g and were divided into 5 groups. The 1 group as positive control group and the 2 group received standard drug Indomethacin at a dose of 10mg/kg per oral. The 3rd, 4th and 5th groups received lecithin 100mg/Kg, 300mg/Kg and 600mg/Kg each respectively by oral route. After 30 min of 0.1mL CFA injection into the sub plantar region of left hind paw on ‘0’ day, Standard Indomethacin (10mg/Kg p.o.) and lecithin were administered orally once daily till 24th day. The antiarthritic effect of the extracts as well as standard evaluate by measuring paw volume of inject paw on every 2days of 24 days study by using Vernier callipers. The mean changes in injected paw volume with respect to initial paw volume were calculated on respective days and % inhibition of paw volume with respect control group was calculated. The changes in body weight record daily. On the day 24th blood was withdrawn from the each the animal through retro-orbital vein puncture by anesthetizing the animal with ether. The blood was collected into vials containing EDTA and the biochemical parameters like haemoglobin content, WBC count, and RBC analysed and animals sacrificed by giving high dose of anesthesia. Left hind paw was dissected and performed histopathology studies.

3. RESULTS

Table 1: Paw volume thickness measured by using Vernier calipers (n=6)

<table>
<thead>
<tr>
<th>Days</th>
<th>Control (mm)</th>
<th>Standard</th>
<th>Lecithin100mg/kg</th>
<th>Lecithin300mg/kg</th>
<th>Lecithin600mg/kg</th>
</tr>
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<tbody>
<tr>
<td>0</td>
<td>30±0.21</td>
<td>28±0.19</td>
<td>32±0.19</td>
<td>32±0.19</td>
<td>33±0.21</td>
</tr>
<tr>
<td>2</td>
<td>32±0.31</td>
<td>25±0.20</td>
<td>31±0.21</td>
<td>30±0.21</td>
<td>29±0.21</td>
</tr>
<tr>
<td>4</td>
<td>31±0.21</td>
<td>22±0.19</td>
<td>30±0.21</td>
<td>29±0.21</td>
<td>30±0.20</td>
</tr>
<tr>
<td>6</td>
<td>34±0.16</td>
<td>20±0.16</td>
<td>28±0.22</td>
<td>27±0.24</td>
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</tr>
<tr>
<td>8</td>
<td>34±0.31</td>
<td>18±0.22</td>
<td>27±0.20</td>
<td>25±0.22</td>
<td>24±0.19</td>
</tr>
<tr>
<td>10</td>
<td>34±0.31</td>
<td>15±0.21</td>
<td>24±0.21</td>
<td>23±0.20</td>
<td>17±0.20</td>
</tr>
<tr>
<td>12</td>
<td>34±0.31</td>
<td>13±0.12</td>
<td>23±0.22</td>
<td>20±0.19</td>
<td>15±0.19</td>
</tr>
<tr>
<td>14</td>
<td>34±0.31</td>
<td>11±0.21</td>
<td>21±0.19</td>
<td>18±0.17</td>
<td>13±0.18</td>
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<tr>
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<td>9±0.18</td>
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<tr>
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<tr>
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<td>33±0.21</td>
<td>5±0.21</td>
<td>17±0.21</td>
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<td>5±0.20</td>
</tr>
<tr>
<td>24</td>
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<td>1±0.21*</td>
<td>15±0.21*</td>
<td>10±0.21*</td>
<td>3±0.21*</td>
</tr>
</tbody>
</table>

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4. CONCLUSION

Rheumatoid arthritis (RA) is a chronic systemic autoimmune inflammatory disease that affects mainly the small joints of the hands and feet. RA is one of the most common inflammatory joint diseases and causes premature mortality, disability and compromised quality of life. RA is widely prevalent throughout the world. The overall worldwide prevalence is 0.8% and steadily increases to 5% in women over the age of 70. RA is two to three times more common in women compared to men. In India the prevalence has been estimated to be 0.7%. Accordingly the main objective of the present invention is to evaluate the activity of lecithin using in-vivo model, on CFA induced rheumatoid arthritis in Wistar rats. This study was approved by IACE under 027/IAEC/NCPCA/M.Pharm/ 2016-17. Wistar rats of weights 250-260 g were selected & divided into 6 groups of 6 each. After the induction of CFA, the animal develops inflammatory responses and shows paw edema.

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